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Dynamics of an SEIR Epidemic Model with Vaccination, Treatment and Saturated Incidence: Threshold Analysis and Numerical Study

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Abstract: Vaccination and treatment are the two main tools used to bring an outbreak under control, yet they act at different stages of the infection and are rarely compared inside a single, simple mathematical framework. In this paper we study a deterministic SEIR model in which susceptible people are vaccinated at a constant rate, infectious people are treated at a constant per-capita rate, and the force of infection saturates at high prevalence to reflect behavioural change and limited contact. We derive the control reproduction number R_c with the next-generation method and show that the disease-free equilibrium is globally asymptotically stable when $R_c < 1$, while a unique endemic equilibrium exists, with an explicit closed form, whenever $R_c > 1$. The model therefore has no backward bifurcation. A normalised sensitivity analysis ranks the transmission rate, vaccination rate and recovery-related rates as the most influential parameters. Numerical simulations with illustrative parameters show that the combined strategy lowers the epidemic peak by about 85% compared with no intervention, and that treatment is the faster-acting measure while vaccination is the more effective long-run one. The results give a compact, analytically tractable basis for comparing intervention mixes.

Keywords: SEIR model; vaccination; treatment; saturated incidence; reproduction number; global stability; sensitivity analysis.

I. INTRODUCTION

Mathematical models have shaped the way we think about infectious disease since Kermack and McKendrick [8] showed, almost a century ago, that a few ordinary differential equations are enough to explain why an epidemic rises, peaks and dies out without exhausting the susceptible population. The compartmental idea they introduced, in which people are grouped by their infection status and moved between groups at given rates, is still the backbone of most modern work [1, 6, 7]. Textbooks such as Anderson and May [1], Keeling and Rohani [7], Brauer et al. [2] and the survey by Hethcote [6] describe how far the approach has travelled: from measles and influenza to HIV, tuberculosis and, more recently, coronavirus disease.

The SEIR model is a natural next step beyond the basic SIR system whenever an infection has a noticeable latent period. People who have been exposed carry the pathogen but are not yet infectious, and ignoring that delay tends to overstate how fast an outbreak grows [7]. For many respiratory infections the latent stage lasts several days, which is long enough to matter for both forecasting and control [1, 6].

Control measures are usually discussed under two headings. Vaccination removes people from the susceptible pool before they meet the pathogen; its effect is preventive and builds up gradually [1, 7]. Treatment, in contrast, acts on people who are already infectious, shortening the time they spend transmitting [2]. Treatment has been studied extensively in its own right, and Wang [12] showed that a limited treatment capacity can even produce a backward bifurcation in an SIR setting, which makes the threshold condition much harder to interpret. Vaccination models with imperfect protection have a similar reputation, as the work of Kribs-Zaleta and Velasco-Hernández [9] illustrates. Because of this, it is useful to know how a model behaves when both measures are present but kept deliberately simple. A second ingredient is the incidence function. The classical bilinear term βSI implies that every infectious person adds to the force of infection without limit. In practice, crowding effects, protective behaviour and finite contact capacity flatten the curve at high prevalence, and Capasso and Serio [3] proposed a saturated form to capture this. We adopt the form $\beta SI/(1 + \alpha I)$, which reduces to the bilinear case when $\alpha = 0$.

The aim of this paper is to bring these three elements together in one model and to answer a few concrete questions. When does the disease die out, and how do the vaccination and treatment rates enter that condition? When it does persist, how large is the endemic level and does saturation change the threshold?

Which parameters matter most? We establish well-posedness, compute the reproduction number, prove global stability of the disease-free state, give an explicit endemic equilibrium, and then illustrate the findings numerically. The remainder of the paper is organised as follows. Section 2 formulates the model. Section 3 treats positivity and boundedness. Sections 4 and 5 deal with equilibria and stability. Section 6 presents the sensitivity analysis, Section 7 the numerical experiments, and Sections 8 and 9 close with a discussion and conclusions.

II. MODEL FORMULATION

The total population at time t is divided into four compartments: susceptible $S(t)$, exposed $E(t)$, infectious $I(t)$ and recovered or immune $R(t)$. Individuals enter the susceptible class at a constant rate Λ and leave every class through natural death at rate μ , as in the standard compartmental setting [6, 7]. The following assumptions are made.

- Susceptible individuals are vaccinated at a constant effective rate ν , which already accounts for coverage and vaccine efficacy. Successfully vaccinated individuals move directly to the recovered class and keep their immunity for life, a simplification commonly made in vaccination models [1, 9].
- Infection occurs through contact with infectious individuals with saturated incidence $\beta SI/(1 + \alpha I)$, where β is the transmission rate and $\alpha \geq 0$ measures the inhibition effect [3].
- Exposed individuals become infectious at rate σ , so $1/\sigma$ is the mean latent period [1].
- Infectious individuals recover naturally at rate γ , die from the disease at the additional rate d , and receive treatment at a constant per-capita rate τ . Treated individuals recover and become immune.

These assumptions lead to the system

$$dS/dt = \Lambda - \beta SI/(1 + \alpha I) - (\mu + \nu)S, \quad (1)$$

$$dE/dt = \beta SI/(1 + \alpha I) - (\mu + \sigma)E, \quad (2)$$

$$dI/dt = \sigma E - (\mu + d + \gamma + \tau)I, \quad (3)$$

$$dR/dt = \nu S + (\gamma + \tau)I - \mu R. \quad (4)$$

with non-negative initial conditions. To shorten the notation we write $a = \mu + \sigma$ and $k = \mu + d + \gamma + \tau$, so that $1/k$ is the mean time an infectious person spends in the infectious class. All parameters are non-negative, and Λ , μ , β , σ and k are strictly positive. Because the first three equations do not involve R , the dynamics of the system are determined by the subsystem (1)–(3), and R can be recovered afterwards from (4).

III. POSITIVITY AND BOUNDEDNESS

A model for populations only makes sense if its solutions stay non-negative and do not blow up [2]. We check both properties.

Theorem 1. *For every non-negative initial condition the solution of system (1)–(4) exists for all $t \geq 0$, remains non-negative, and is bounded. The region $\Gamma = \{(S, E, I, R) \in \mathbb{R}_+^4 : S + E + I + R \leq \Lambda/\mu\}$ is positively invariant and attracts all solutions.*

Proof. Look at the vector field on the boundary of the positive orthant. When $S = 0$, we have $dS/dt = \Lambda > 0$. When $E = 0$, $dE/dt = \beta SI/(1 + \alpha I) \geq 0$. When $I = 0$, $dI/dt = \sigma E \geq 0$, and when $R = 0$, $dR/dt = \nu S + (\gamma + \tau)I \geq 0$. In every case the vector field points into the orthant or is tangent to it, so non-negative solutions stay non-negative. Let $N = S + E + I + R$. Adding the four equations gives $dN/dt = \Lambda - \mu N - dI \leq \Lambda - \mu N$, and hence $N(t) \leq \Lambda/\mu + (N(0) - \Lambda/\mu)e^{-\mu t}$. Therefore N is bounded, $\limsup N(t) \leq \Lambda/\mu$, and the solution can be continued for all time. If $N(0) \leq \Lambda/\mu$, then $N(t) \leq \Lambda/\mu$ for all t , which proves invariance of Γ . ■

From now on we restrict attention to Γ . A further consequence of the first equation is useful later. Since the incidence term is non-negative, $dS/dt \leq \Lambda - (\mu + \nu)S$, and a standard comparison argument [2] gives $\limsup S(t) \leq S_0$, where $S_0 = \Lambda/(\mu + \nu)$ is defined in the next section.

IV. EQUILIBRIA AND THE REPRODUCTION NUMBER

A. Disease-free equilibrium and reproduction number

Setting $E = I = 0$ in the equilibrium equations gives the disease-free equilibrium

$$DFE = (S_0, 0, 0, R_0'), \quad S_0 = \Lambda/(\mu + \nu), \quad R_0' = \nu \Lambda/(\mu(\mu + \nu)). \quad (5)$$

Vaccination lowers the susceptible population at the disease-free state from Λ/μ to $\Lambda/(\mu + \nu)$, and moves the difference into the immune class. To find the threshold quantity we apply the next-generation method of Diekmann et al. [5] and van den Driessche and Watmough [11] to the infected compartments (E, I) . The matrix of new infections F and the matrix of transitions V , both evaluated at the DFE, are

$$F = [[0, \beta S_0], [0, 0]], \quad V = [[a, 0], [-\sigma, k]]. \quad (6)$$

Computing V^{-1} and multiplying gives the next-generation matrix FV^{-1} , whose only non-zero eigenvalue is its spectral radius. The control reproduction number is therefore

$$R_c = \beta\sigma S_0 / ((\mu + \sigma)(\mu + d + \gamma + \tau)) = \beta\sigma\Lambda / ((\mu + \nu)(\mu + \sigma)(\mu + d + \gamma + \tau)). \quad (7)$$

The expression has a clear biological reading, in line with the general interpretation of reproduction numbers in [5, 11]. The factor $\sigma/(\mu + \sigma)$ is the probability that an exposed person survives the latent period and becomes infectious, βS_0 is the rate at which an infectious person infects a fully susceptible population at the disease-free state, and $1/k$ is the average infectious period. The saturation parameter α does not appear in R_c , because at the start of an outbreak I is close to zero and the incidence is effectively bilinear. Without any intervention ($\nu = \tau = 0$) the expression reduces to the basic reproduction number $R_0 = \beta\sigma\Lambda / (\mu(\mu + \sigma)(\mu + d + \gamma))$ [5, 11].

Solving $R_c = 1$ for the intervention parameters gives the critical values

$$\nu_c = \beta\sigma\Lambda / ((\mu + \sigma)k) - \mu, \quad \tau_c = \beta\sigma\Lambda / ((\mu + \nu)(\mu + \sigma)) - (\mu + d + \gamma). \quad (8)$$

Any vaccination rate above ν_c (for a given τ), or any treatment rate above τ_c (for a given ν), brings the control reproduction number below one. Note that ν_c falls as τ rises, so the two measures substitute for each other; this is quantified in Section 7.

B. Endemic equilibrium

Theorem 2. *System (1)–(4) always has the disease-free equilibrium. If $R_c \leq 1$ there is no other equilibrium in Γ . If $R_c > 1$ there is exactly one endemic equilibrium (S^*, E^*, I^*, R^*) , given by $I^* = (\mu + \nu)(R_c - 1) / (\beta + \alpha(\mu + \nu))$, $S^* = S_0(1 + \alpha I^*) / R_c$, $E^* = kI^* / \sigma$, and $R^* = (\nu S^* + (\gamma + \tau)I^*) / \mu$.*

Proof. Let $I^* > 0$ be the infectious component of an equilibrium. From $dI/dt = 0$ we get $E^* = kI^* / \sigma$. Substituting this into $dE/dt = 0$ yields $\beta S^* / (1 + \alpha I^*) = ak / \sigma$, which can be rearranged as $S^* = S_0(1 + \alpha I^*) / R_c$, using the definition (7). Adding the equations for S and E at equilibrium gives $\Lambda - (\mu + \nu)S^* - \alpha E^* = 0$. Since $\Lambda = (\mu + \nu)S_0$, this reads $(\mu + \nu)(S_0 - S^*) = \alpha kI^* / \sigma$. Now $ak / \sigma = \beta S_0 / R_c$, so after substituting S^* and cancelling the common factor S_0 / R_c we are left with $(\mu + \nu)(R_c - 1 - \alpha I^*) = \beta I^*$. This is linear in I^* , and its unique solution is the expression stated above. It is positive if and only if $R_c > 1$. The values of S^* , E^* , R^* then follow directly from the remaining equations and are positive. ■

Two features of this result are worth pointing out. First, the endemic equilibrium can be written in closed form, which is unusual for models with a saturated incidence, and it is unique. Because the equilibrium appears only when R_c crosses one from below and is then unique and increasing in R_c , the bifurcation at $R_c = 1$ is forward: the model cannot display the backward bifurcation and bistability reported for some treatment and vaccination models [9, 12]. In practical terms, pushing R_c below one is both necessary and sufficient to eliminate the disease. Second, the saturation parameter appears only in the denominator of I^* . Increasing α therefore lowers the endemic level but leaves the threshold unchanged, so saturation reduces the burden of an established epidemic without making elimination any easier.

V. STABILITY ANALYSIS

Theorem 3 (local stability). *The disease-free equilibrium is locally asymptotically stable if $R_c < 1$ and unstable if $R_c > 1$.*

Proof. The Jacobian matrix at the DFE is block triangular. Its eigenvalues are $-(\mu + \nu)$, $-\mu$, and the two eigenvalues of the matrix $F - V = [[-a, \beta S_0], [\sigma, -k]]$, which satisfy $\lambda^2 + (a + k)\lambda + ak(1 - R_c) = 0$. The trace $-(a + k)$ is always negative. The determinant is $ak(1 - R_c)$, which is positive when $R_c < 1$, so both eigenvalues have negative real part. When $R_c > 1$ the determinant is negative, so there is one positive real eigenvalue and the equilibrium is unstable. ■

Local stability only says what happens for small perturbations. For public health purposes we want to know that the disease dies out from any initial state, and that is what the next result provides.

Theorem 4 (global stability). *If $R_c < 1$, the disease-free equilibrium is globally asymptotically stable in Γ .*

Proof. Define the function $L = E + (a/\sigma)I$, which is non-negative and vanishes only when $E = I = 0$. Differentiating along solutions and using (2) and (3), $dL/dt = \beta SI / (1 + \alpha I) - aE + (a/\sigma)(\sigma E - kI) = \beta SI / (1 + \alpha I) - (ak/\sigma)I$. The incidence satisfies $\beta SI / (1 + \alpha I) \leq \beta SI$, so $dL/dt \leq (ak/\sigma)I(\beta\sigma S / (ak) - 1)$. By the remark at the end of Section 3, for any $\varepsilon > 0$ we have $S(t) \leq S_0 + \varepsilon$ after some finite time. Because $R_c < 1$ we can choose ε small enough that $\beta\sigma(S_0 + \varepsilon) / (ak) < 1$, and then $dL/dt \leq -cI$ for some $c > 0$ and all large t . Thus L is non-increasing, and the largest invariant set on which $dL/dt = 0$ requires $I = 0$, which in turn forces $E = 0$ through equation (3). On this set the equation for S becomes linear and gives $S \rightarrow S_0$, and then $R \rightarrow \nu S_0 / \mu$. By LaSalle's invariance principle [10], every solution in Γ converges to the DFE. Together with Theorem 3 this gives global asymptotic stability. ■

Theorem 4 is the central analytical result of the paper. It says that if vaccination and treatment together push the control reproduction number below one, the disease will disappear regardless of how many people are initially infected. For $R_c > 1$ the disease-free state is unstable and the numerical experiments in Section 7 indicate that solutions settle on the endemic equilibrium of Theorem 2. A rigorous proof of the global stability of the endemic equilibrium would require a more delicate Lyapunov construction, and we leave it for future work.

VI. SENSITIVITY ANALYSIS

To judge which parameters deserve most attention from policy makers we compute the normalised forward sensitivity index of R_c with respect to each parameter p , defined as $Y_p = (\partial R_c / \partial p)(p/R_c)$ [4]. This number tells us by what percentage R_c changes when p is changed by one percent. From (7) the indices for the parameters that appear as simple factors are available in closed form: $Y_\beta = Y_\Lambda = 1$, $Y_\sigma = \mu/(\mu + \sigma)$, $Y_\nu = -\nu/(\mu + \nu)$, and $Y_\tau = -\tau/k$, with Y_γ and Y_d obtained in the same way. The index for μ was evaluated numerically since μ appears in three factors. Table 1 lists the values at the parameter set used in Section 7 with $\nu = 0.02$ and $\tau = 0.10$.

Table 1. Normalised forward sensitivity indices of R_c .

Parameter	Index	Interpretation
β, Λ	+1.00	Proportional increase in R_c
ν	-0.67	Strong reduction through vaccination
τ	-0.45	Moderate reduction through treatment
γ	-0.45	Faster natural recovery shortens infectiousness
μ	-0.43	Higher turnover shrinks the susceptible pool
σ	+0.05	Almost no effect, latent period is short
d	-0.05	Small effect, disease mortality is low

Several things stand out. The transmission rate and the recruitment rate have the largest positive effect, and since a 10% reduction in contacts (for example through distancing) cuts R_c by exactly 10%, non-pharmaceutical measures remain valuable alongside vaccination and treatment. Among the controls, vaccination has the largest negative index at these values. This is partly because $\nu = 0.02$ is large relative to the natural turnover rate $\mu = 0.01$, so that the vaccinated fraction at the disease-free state is substantial. The treatment index is exactly equal in magnitude to that of the natural recovery rate, which reflects the fact that both just shorten the infectious period in the same additive way.

VII. NUMERICAL SIMULATIONS

We now illustrate the analytical results. The parameter values in Table 2 are chosen to be plausible for an acute respiratory infection with a latent period of about five days and an infectious period of about ten; they are of the order commonly used in textbook treatments [1, 7] but are illustrative and are not fitted to any particular data set. Time is measured in days. Equations (1)–(4) were integrated with an adaptive Runge–Kutta scheme (relative tolerance 10^{-9}) in Python, using the initial condition $(S, E, I, R) = (980, 10, 10, 0)$, so that the total population starts at its equilibrium size $\Lambda/\mu = 1000$.

Table 2. Parameter values used in the simulations.

Symbol	Meaning	Value	Unit
Λ	Recruitment rate	10	day ⁻¹
μ	Natural death rate	0.01	day ⁻¹
β	Transmission rate	0.0004	day ⁻¹
α	Saturation (inhibition) parameter	0.01	—
σ	Rate of progression from exposed to infectious	0.2	day ⁻¹

Symbol	Meaning	Value	Unit
γ	Natural recovery rate	0.1	day ⁻¹
d	Disease-induced death rate	0.01	day ⁻¹
ν	Effective vaccination rate	0 – 0.06	day ⁻¹
τ	Treatment rate	0 – 0.30	day ⁻¹

A. Comparison of intervention strategies

Four scenarios were compared: no intervention, vaccination only ($\nu = 0.02$), treatment only ($\tau = 0.10$), and both measures together. The resulting control reproduction numbers and epidemic summaries are given in Table 3, and the time course of the infectious population is plotted in Figure 1.

Table 3. Reproduction numbers and epidemic summaries for the four scenarios (400-day horizon).

Scenario	R_c	Peak I	Peak day	$I(400)$	I^* (theory)
No intervention	3.17	97.4	45.6	43.5	43.5
Vaccination only ($\nu = 0.02$)	1.06	49.3	33.8	3.03	2.49
Treatment only ($\tau = 0.10$)	1.73	31.2	48.8	14.7	14.6
Vaccination + treatment	0.58	14.8	20.2	≈ 0	0

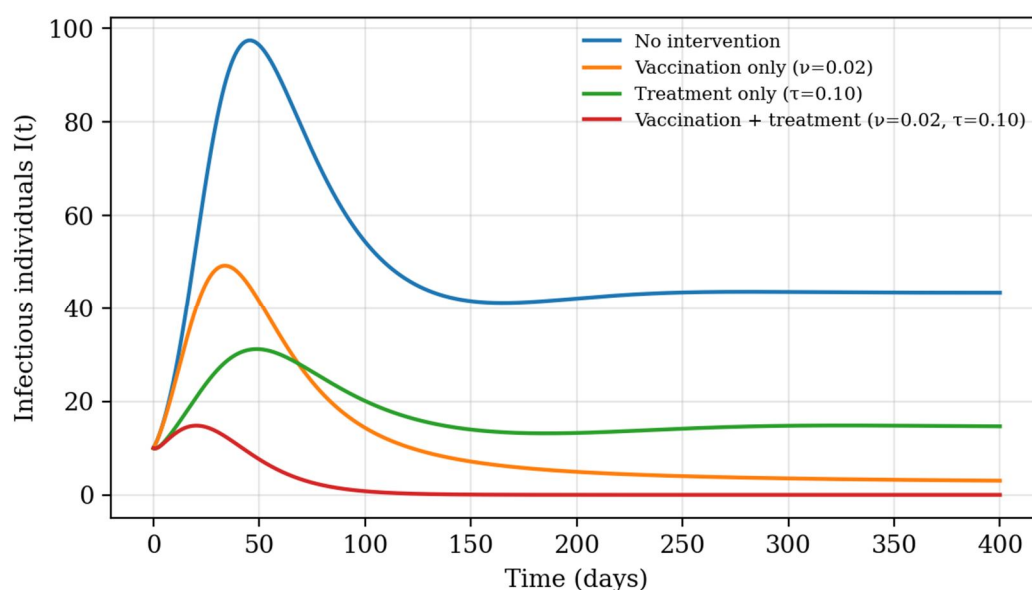


Figure 1. Number of infectious individuals under the four intervention scenarios.

Without intervention, $R_c = 3.17$ and the epidemic peaks at about 97 infectious individuals around day 46. The solution then oscillates slightly and settles at the endemic level of 43.5, in agreement with the value predicted by Theorem 2 (43.49). The full set of compartments for this baseline case is shown in Figure 2: the susceptible pool is depleted quickly, the recovered class grows to roughly 435, and the infection never fully disappears because new susceptibles keep arriving through the recruitment term.

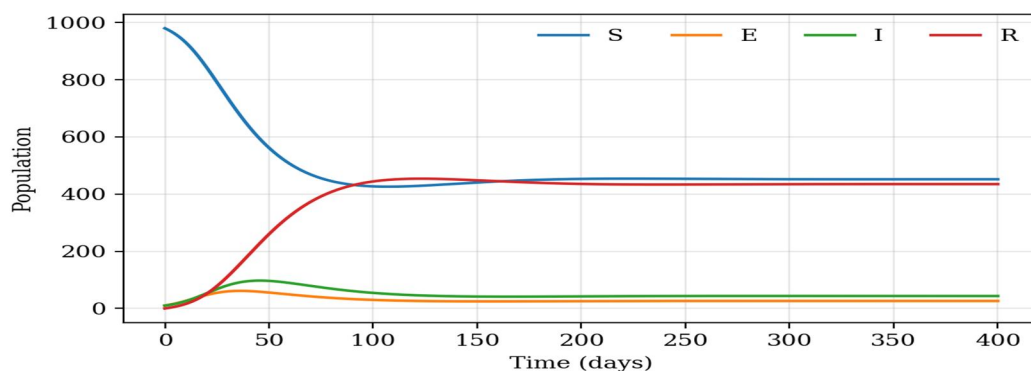


Figure 2. Evolution of the four compartments without intervention ($R_c = 3.17$).

Both single measures reduce the burden, but in different ways. Treatment alone reduces the peak by roughly 68%, from 97.4 to 31.2, even though $R_c = 1.73$ remains well above one. Vaccination alone at $v = 0.02$ lowers the peak by only about 49%, but it brings R_c down to 1.06, very close to the threshold, which is why the long-run endemic level drops from 43.5 to about 2.5. The reason for this difference is timing. Treatment acts immediately on those who are already infectious, whereas vaccination needs time to deplete the susceptible pool, so its benefits show up mostly after the first wave. The slight gap between $I(400) = 3.03$ and the theoretical 2.49 in the vaccination-only row is also a timing effect: when R_c is this close to one the approach to equilibrium is slow, and the solution has not fully converged by day 400. The combined strategy is clearly the strongest. It gives $R_c = 0.58$, cuts the peak by about 85% to 14.8 and moves it forward to day 20, after which the infection decays to essentially zero, as Theorem 4 guarantees. The combined effect is also larger than a simple sum of the individual effects in terms of R_c : because the two measures multiply rather than add in (7), each one gets more out of the other.

B. Intervention thresholds

Figure 3 shows the value of R_c over the (v, τ) plane, with the white curve marking the elimination threshold $R_c = 1$. With no treatment, the vaccination rate must exceed $v_c = 0.0217$ per day for the disease to die out. If treatment is also provided at $\tau = 0.05$ the requirement falls to 0.0124, and at $\tau = 0.10$ to only 0.0073. Without vaccination, treatment alone would have to reach $\tau_c = 0.261$ per day, which means treating a quarter of the infectious population every day and is unlikely to be feasible for a long period. The figure therefore makes the trade-off concrete: a moderate investment in both measures achieves what a very large investment in either one alone would be needed for.

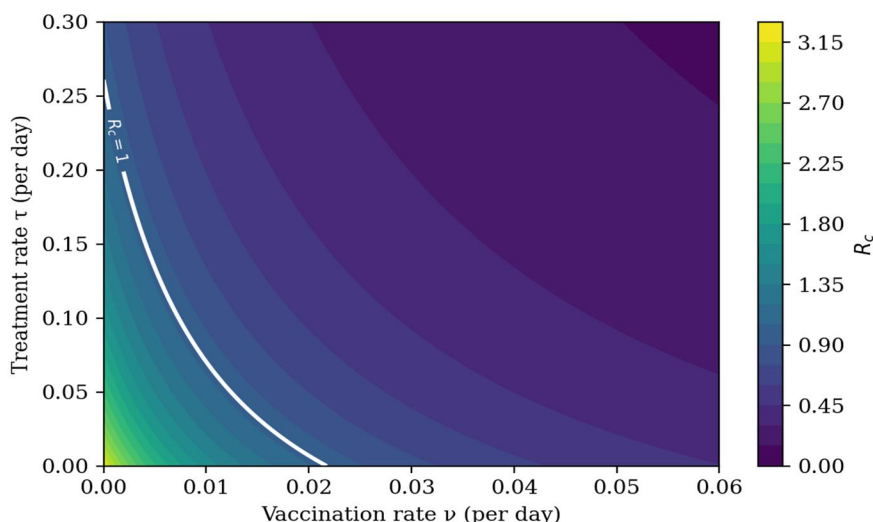


Figure 3. Contour plot of the control reproduction number R_c as a function of the vaccination rate v and the treatment rate τ . The white curve is $R_c = 1$.

C. Effect of the vaccination rate on the peak

To see how much additional vaccination is worth once treatment is in place, we fixed $\tau = 0.05$ and varied ν from 0 to 0.06. The resulting epidemic peak is plotted in Figure 4. The peak falls from 55.3 at $\nu = 0$ to 25.6 at $\nu = 0.02$, and then to 15.6 at $\nu = 0.06$. Most of the benefit therefore comes from the first few increments of the vaccination rate; going from 0.02 to 0.06 reduces the peak by a further ten individuals, while the first 0.02 removed about thirty. This diminishing-returns pattern is useful when deciding how far to push vaccination coverage in a resource-limited setting.

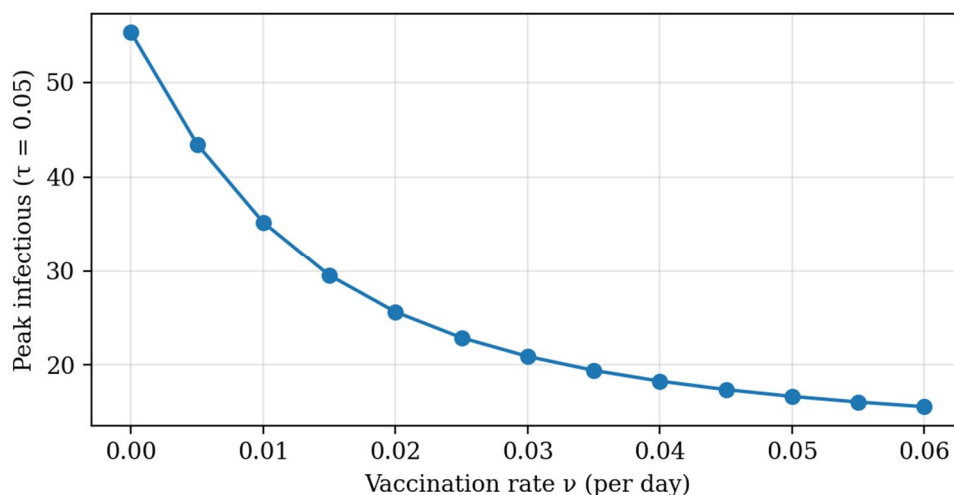


Figure 4. Epidemic peak as a function of the vaccination rate for a fixed treatment rate $\tau = 0.05$.

VIII. DISCUSSION

The model studied here is deliberately simple, and that simplicity is what allows a complete threshold analysis. The results can be summarised in three messages. The first is that the question of elimination reduces to one inequality, $R_c < 1$, with no hidden bistability, so a public health authority can use (7) and (8) directly to set targets, in line with the classical threshold idea [6, 11]. The second is that vaccination and treatment are complements, not rivals. They enter the reproduction number multiplicatively, and the numerical results show that treatment protects well in the short run while vaccination secures the long run. The third is that saturation of the incidence changes the size of an endemic epidemic but not the threshold, so behavioural effects should not be mistaken for a substitute for control.

There are several limitations. Vaccination is assumed to give permanent and complete protection, which is not realistic for many vaccines; waning immunity and partial efficacy would add a vaccinated compartment and are known to complicate the threshold behaviour [9]. Treatment is taken to be proportional to the number of infectious people, so the model cannot capture a limited capacity such as a fixed number of hospital beds, which is exactly the situation in which Wang [12] found backward bifurcation. Demographic change is minimal, with no age structure, and the parameters are illustrative rather than estimated. Finally, global stability of the endemic equilibrium has been supported numerically but not proved.

These limitations suggest natural extensions: adding a vaccinated class with waning protection, replacing the linear treatment term by a saturated one, introducing a time delay for the latent period, or replacing the integer-order derivatives with fractional-order operators to represent memory effects. Fitting the model to surveillance data would also allow the intervention thresholds to be estimated for a specific disease.

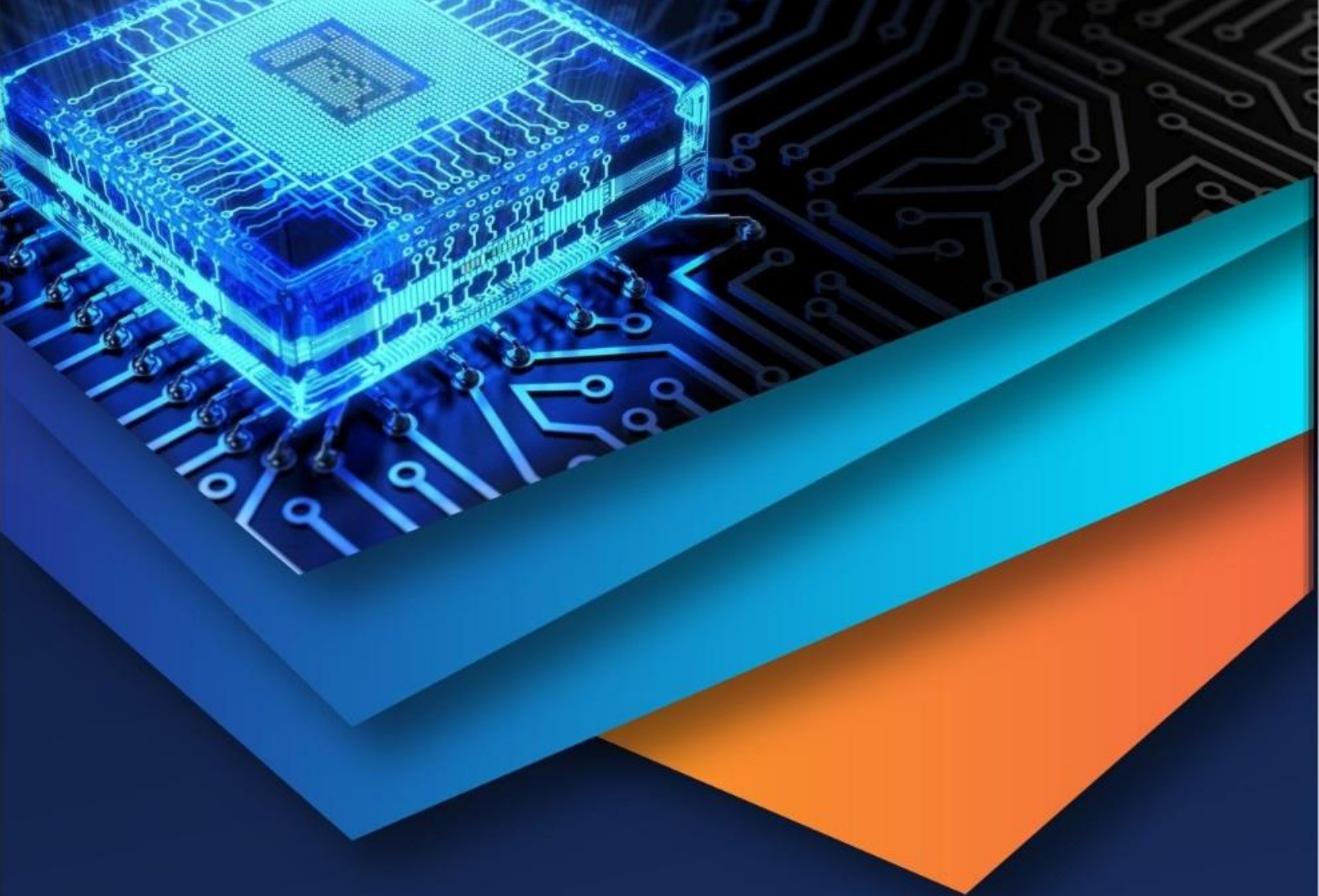
IX. CONCLUSION

We have analysed an SEIR model with constant-rate vaccination, constant per-capita treatment and a saturated incidence rate. The model is well posed on a biologically feasible region. Its control reproduction number R_c is given in closed form, the disease-free equilibrium is globally asymptotically stable for $R_c < 1$, and a unique endemic equilibrium with an explicit expression exists for $R_c > 1$, which excludes backward bifurcation. Sensitivity analysis highlights the transmission rate, the vaccination rate and the recovery-related rates as the most influential parameters. In the numerical experiments, combining vaccination with treatment reduced the epidemic peak by about 85% and drove the infection to extinction, whereas either measure on its own left the disease endemic. The analysis supports the use of mixed strategies and provides explicit threshold formulas that can be used to size them.



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